The initial clinical impression of viral infection with exanthem was challenged when the child's mother phoned the following day and reported the development of erythema of the palms and soles and a strawberry tongue. Desquamation of the palms, soles and fingers ten days after the initial examination in association with leukocytosis, thrombocytosis and an elevated erythrocyte sedimentation rate confirmed the diagnosis of Kawasaki syndrome.

Although both of these children had moderate malaise and anorexia during the acute phase of their illness, hospital admission was never considered necessary for either diagnostic, therapeutic or social purposes. The diagnosis of Kawasaki syndrome was apparent during the subacute phase in one patient and during the acute phase in the other. In retrospect, bulbar conjunctivitis without discharge and erythema of palms and soles were clues to the correct diagnosis early in the course of the disease when fever and rash were nonspecific presenting signs.

These two cases, which were both seen by one pediatrician in the first year of a new practice, occurred over a ten-month period. Although the children lived in the same region of an urban community, their socioeconomic status was different and they did not share preschools or play areas. A retrospective, informal survey of several group pediatric practices and a large health maintenance organization in the same community did not reveal a single case of Kawasaki syndrome without admission to hospital diagnosed during the same period of time. The number of children admitted to hospital with Kawasaki syndrome during this period of time was not higher than in previous years.

This report suggests that Kawasaki syndrome may be masquerading as other diagnoses in pediatric office practices. Careful examination of the conjunctiva, palms, soles and lips of young children who present with prolonged fever and a nonspecific generalized rash may lead to the discovery of many more patients with less severe forms of Kawasaki syndrome than previously reported. If this assumption is correct, the increased number of cases may provide clinical investigators with new clues to the cause of this elusive syndrome.

Summary

Published clinical, epidemiologic and laboratory data on Kawasaki syndrome have left primary care pediatricians with the impression that these children usually are acutely ill with a toxic presentation. Two cases from one general pediatric practice suggest that early symptoms and signs of this syndrome may be mild, atypical and resemble those of other, more common diseases. As coronary artery aneurysms and thrombosis may develop in children with variable expressions of Kawasaki syndrome, primary care pediatricians should be aware of the milder presentations that may be seen in an office practice.

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Community-Acquired *Pseudomonas aeruginosa* Pneumonia Associated With the Use of a Home Humidifier

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Pseudomonas aeruginosa is well recognized as a nosocomial pathogen. In an infection surveillance study at the Boston City Hospital in 1970, this organism accounted for 18% of postoperative wound infections, 17% of nosocomial lower respiratory tract infections and 11% of hospital-acquired urinary tract infections.[1] However, none of the community-acquired pneumonias or urinary tract infections in this study were due to *P aeruginosa*. In two prospective studies of cases of community-acquired pneumonia, Gram-negative bacteria were documented as the etiologic agents in 16%[2] and 20%[3] of cases. Of the 719 patients in these two studies, only 2 had *P aeruginosa* identified in their sputum specimens and none from blood specimens. When community-acquired *P aeruginosa* pneumonia does occur, it is usually in patients with severe underlying diseases or in parenteral drug abusers. Its occurrence in previously healthy persons is extremely rare.[4]

We report the case of a man with a 25-year history of asthma who had community-onset *P aeruginosa* pneumonia in an unusual epidemiologic setting. Other sources of community-onset *Pseudomonas* infections are discussed.

Report of a Case

A 39-year-old man presented to the emergency room with shortness of breath. Five days before admission he noticed the onset of cough productive of thick yel-

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low sputum. For two days before admission he had had many episodes of rigors and drenching sweats. Pleuritic pain was localized to the left anterior thorax.

The patient's medical history included 25 years of asthma. During the several years before admission he had had severe symptoms year-round. Treatment was obtained at several outpatient clinics and emergency rooms. There were no hospital admissions during the 12 months preceding this admission. Medications included Tedral (a combination product containing ephedrine hydrochloride, phenobarbital and theophylline), Elixophyllin (theophylline), guaifenesin (Robitussin) and isoproterenol hydrochloride (Isuprel Mistometer). The patient had been using a home humidifier for the six months before admission.

On physical examination he was obviously chronically ill, asthenic and in acute respiratory distress. Temperature was 40.3°C (104.6°F) rectally, blood pressure 88/60 mm of mercury, pulse 140 per minute and regular and respiratory rate 40 per minute. Diffuse wheezes were present and breath sounds were decreased at the left base. The patient required emergency intubation and was transferred to the medical intensive care unit. Specimens of blood, urine and sputum were obtained for culture and were examined for the presence of aerobic and anaerobic bacteria and mycobacteria. Initial antibiotic therapy included ampicillin, gentamicin sulfate and clindamycin.

Laboratory data included a hemoglobin of 13.5 grams per dl with a hematocrit of 49%. The leukocyte count was 28,000 per μl. Arterial blood gas determinations done while the patient was breathing room air showed a partial oxygen pressure of 80 mm of mercury, a partial carbon dioxide pressure of 25 mm of mercury and a pH of 7.49. A chest x-ray study showed a left lower lobe infiltrate and small pleural effusion. A sputum Gram's stain showed heavy leukocytes with many Gram-negative rods. No intracellular organisms were seen. An acid-fast stain was negative.

Cultures of sputum, pleural fluid and blood specimens were positive for *P aeruginosa*. No other pathogen was identified. The patient's purified-protein derivative test was negative. Serum immunoglobulins and α_1-antitrypsin levels were normal. The antibiotic regimen was changed to carbenicillin (24 grams a day) and gentamicin (300 mg a day). On this regimen his condition slowly improved. His course was complicated by a bronchopleural fistula requiring surgical decortication. He was discharged from hospital 53 days after admission, having received antipseudomonad therapy for 46 days. Four months later he was readmitted to hospital with *Hemophilus influenzae* pneumonia. *P aeruginosa* was not present in his sputum at this time or 14 months later when he died of tuberculosis.

Discussion

The identification of *Pseudomonas aeruginosa* as the etiologic agent prompted a review of the patient's history for possible exposures. There were no recent admissions to hospital, antibiotic usage, parenteral drug use, trauma or use of whirlpool baths. However, for the past five months the patient had been using a home humidifier. The patient's home was visited three days after his admission to the hospital. The humidifier was in the bathtub, covered with dust and contained water. Cockroaches and fleas were observed both within the unit and on the surface. Cultures of the bathtub water trap, as well as the humidifier rim, plunger and water were all positive for *P aeruginosa*. All home and patient isolates had identical patterns of antibiotic sensitivities and pyocin typing.*

P aeruginosa has been isolated from 2% of pharyngeal cultures of persons not admitted to hospital.[5] Like other pseudomonads, this most common member of the Pseudomonadaceae is ubiquitous in water and moist soil. However, infection is seen in only a few unique settings. Outbreaks of *P aeruginosa* folliculitis and cellulitis have been linked to the use of whirlpools and hot tubs in various motels and spas.[6] Community-acquired endocarditis is most commonly seen in persons with heroin addiction where the organism may be transmitted by using contaminated syringes.[7]

P aeruginosa as a cause of community-acquired pneumonia is very rare.[1,2] When seen, it is usually in patients with cystic fibrosis or severe combined immunodeficiency syndrome and chronic granulomatous disease. *Staphylococcus aureus, Streptococcus pneumoniae* and *H influenzae* are frequent antecedent pathogens before colonization and infection with pseudomonads, which are usually mucoid and nontypable. Recurrent and ongoing courses of antibiotics and the use of home humidifiers are frequently a part of the therapeutic regimen of these patients. Our patient had none of the predisposing diseases associated with community infection with *P aeruginosa* but had frequently used a humidifier.

Contaminated anesthesia machines, mechanical ventilators, inhalation therapy equipment, delivery room resuscitators and humidifiers have all been implicated in hospital-acquired respiratory tract diseases.[8,9] Humidification connotes the addition of moisture to the air without production of aerosols. Portable humidifiers with a spinning disc, similar to that used by our patient, are more appropriately considered fine-particle-reservoir nebulizers and have been a source of *P aeruginosa* pulmonary infections in hospital.[10] Their home use has been associated with fungal aerosols, hypersensitivity pneumonitis and asthma.[11] However, no community-acquired cases of Gram-negative pneumonia have previously been traced to a contaminated respiratory device in the home. Unlike other home respiratory devices, patients do not come in direct contact with humidifiers of this type. Coupled with the ubiquitousness of *P aeruginosa* in water, its presence in the bathtub water trap of our patient and the absence of clinical conditions known to be associated with colonization with pseudomonads, it is far more likely

*Pyocin typing was done by H. Griebel, MD, and T. Bird, PhD.

that a contaminated machine led to colonization and subsequent infection in our patient than that the infected patient directly contaminated his machine.

Maintenance of our patient's humidifier was certainly substandard. Stagnation of water for long periods of time would permit a pronounced increase in colony counts available for aerosolization. Respiratory devices such as the one used by our patient are used for "humidification" in hospitals and homes throughout the country without proved therapeutic efficacy. When efficacy is questionable, utter safety becomes the overriding concern. Patients such as ours point up the possibility for a rare but potentially life-threatening complication. We suggest that if these portable home humidifiers are used, patients should be instructed to discard residual water after each use, dust and rinse thoroughly before use after storage and only fill with water just prior to usage.

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